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GERIATRICS

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Guest Editorial

RORY H. G. FISHER, B.A., M.B., Ch.B., M.R.C.P.(Ed.), F.R.C.P.(C)

Dr. Fisher attended Dublin University, where he received his B.A. in 1957 and his M.B., Ch.B. in 1959. He served as Senior Registrar in Internal Medicine and Geriatrics, Department of Medicine in The London Hospital, London, England from 1967 to 1970. His present appointments are many. In addition to his position as head of the Department of Extended Care at Sunnybrook Medical Centre, he holds an appointment in the Faculty of Medicine at the University of Toronto as assistant professor in the Department of Family and Community Medicine, where he is also Co-ordinator of Chronic and Geriatric Care. He is on the active staff in the Division of Respiratory Disease and General Medicine at Sunnybrook Medical Centre. Dr. Fisher has published several articles on the medical problems of the elderly, the latest of which is "The Day Hospital: In-Patient Alternative" in the Ontario Medical Review, 41, 1974.

Why Geriatrics?

The World Health Organization has defined geriatics as a branch of gerontology and medicine that is concerned with health of the elderly in all its aspects – preventive, clinical, remedial, rehabilitative and continuous surveillance.

Eight per cent of the population of Canada is now over the age of 65, and by the turn of the century this will have risen to 11 per cent. The elderly are now major users of the health care system, and will become increasingly so in the future. In addition, there is an increasing amount of knowledge relative to the biological processes of aging. The medical problems of the aged differ from younger groups with the prevalence of multiple pathology, different presentation and response to disease, differing response to drugs, difficulty of ascertainment of disease, and problems of death and dying.

In spite of the aged population's numerical importance there has, as yet, been little in the way of formal training for health professionals in the special needs of this group. In other countries it has been shown that the elderly may be helped by the formation of geriatric programs in which care is provided by multidisciplinary teams, with each member contributing his or her own skill to the care of the aged. Following these examples, the Department of Extended Care at Sunnybrook Medical Centre has been divided into units to provide different modalities of care. These units include an Assessment Unit, where a full evaluation of every aspect of the patient's problem is made by a multidisciplinary team and a realistic therapeutic program outlined, and a Restorative Unit, where patients' rehabilitation needs in the broadest sense of the word are dealt with. There is also a Psychogeriatric Unit, since it is now well demonstrated that the psychiatric problems of the elderly are amenable to treatment. Long-Stay wards are, of course, necessary in view of the inevitability of many elderly patients being unable to function in the community, but efforts are made to achieve their maximum level of function, to provide an optimal quality of life. The Department's Day Hospital is an extremely valuable means of maintaining patients in the community, by leading to an earlier discharge of patients, bridging the gap between the hospital and the home by carrying out a therapeutic program on an Out-Patient basis. All services place an emphasis on measures aimed at maintaining the elderly in the community.

One problem of health care for the aged has been the negative attitudes of some health professionals. There are, fortunately, signs to suggest this situation is changing, and hopefully with improved education there will be increased understanding of the success that may be achieved in treating the elderly. Although cure may not be so likely, improvement of function with return home may be achieved in many cases. It is important to concentrate on overcoming disabilities rather than curing diseases. Chronological age should be ignored, and the patient assessed on his biological condition. It is indeed just as discriminatory to label a patient by his age as by his racial or religious background. There are many physically and mentally alert patients in their eighties while there are patients in their fifties who are severely handicapped. It should also be realized that the elderly wish to remain independent in the community for as long as possible and that they should be assisted in this aim.

The practice of geriatrics provides a wide variety of interesting clinical conditions and an opportunity to gain experience in a field which is growing in importance, and in which there will be increasing professional opportunities. The awakening concern in geriatrics and gerontology is evidenced by the formation in recent years of the Canadian Association of Gerontology, the Ontario Psychogeriatric Association and the Gerontological Nursing Association. At a university level, in Ontario, the University of Western Ontario has appointed a co-ordinator of geriatric care, McMaster University has a physician responsible for gerontological education in the health sciences, and Queen's University has set up a division of geriatric continuing care medicine. Within the University of Toronto there is the Department of Extended Care at Sunnybrook, with University physicians also attending the well developed geriatric centre at Baycrest, the active geriatric unit at the Toronto Western, and other centres such as Riverdale and the Queen Elizabeth Hospitals.

Various levels of government are becoming more aware of the geriatric problem. In this respect, it is to be noted that in *A New Perspective on the Health of Canadians*, the Federal Ministry of Health proposed the development and support of professional training in gerontology and geriatrics for physicians, nurses and health personnel.

Because of their numbers and needs, the elderly will place a great strain on health services in the future. It therefore follows that more geriatric programs and further education in geriatrics and gerontology will be necessary.

Editorial

In this final issue of Volume LIII of the *Journal* it is appropriate that we express our sincere appreciation to the many who have given generously of their time and abilities over the past year.

We extend our congratulations to the associate editors for the fine issues they produced. Special thanks also to the students of the Art as Applied to Medicine department, whose skillful talents added so much to our publication this year.

Finally, we wish to thank our advertisers, subscribers and patrons for their kind support. In particular, we gratefully acknowledge the generous gifts of Dr. D. Vaughan of San Francisco and the Medical Alumni Association of the University of Toronto.

Our association with the *Journal* has indeed been both pleasant and rewarding, and we wish the editors and staff of the 1976-77 academic year every success.

MICHAEL HOWCROFT
BEVERLY MORNINGSTAR

PUBLICATION GUIDELINES:

1) All articles intended for publication should be submitted to the editor before the deadline which is usually **one** month before the release of the Journal, i.e. for the November 15 issue, the deadline is October 15.
2) Submitted articles should be typed (or legibly written) double-spaced, single sided on paper 8½" x 11" (letter size) and they should not be more than 15 to 20 pages (3,000 to 4,000 words).
3) Illustrations, graphs, diagrams, figures and photographs should be put on a separate sheet and identified by Arabic numerals. A list of explanatory legends for all the figures and diagrams should be put on another sheet. Do not write on the back of photographs.
4) References should be checked with the original sources, designated by Arabic numerals in the text, numbered in order of appearance and put on a separate sheet. Each periodical reference should include in order: the author's name and initials, title of the article, journal name, volume number, page number and year (e.g. 1. ROSEN HJ: Lumbar intervertebral disc surgery: review of 300 cases. Can Med Assoc J. 101: 317, 1969)
5) The editor and associate editors reserve the right to make such editorial changes as are considered necessary to ensure correctness of grammar and spelling, clarification of statements and conformity to Journal style.
6) Acceptance for publication is subject to the understanding that such material is submitted solely to the Journal, and will not be reprinted without the consent of both the editor and author.

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Aging as a Physiological and Biochemical Process

Anne R. Crowe, 7T9

The phenomenon of aging is an intrinsic part of human life and, linked as it is to death, must have been part of man's earliest speculations. Certainly mythology and folk tales endow the aged, if only for their apparent defiance of death, with very special abilities. In historical times the ancient Greek philosophers described the changes that accompany increasing age and discussed their significance. During the middle ages the pursuit of the elixir of life and the philosopher's stone incidentally resulted in the foundation of modern chemistry and physics. Indeed, even today articles about miracle treatments that prevent or reverse the normal effects of time are used to sell popular magazines.

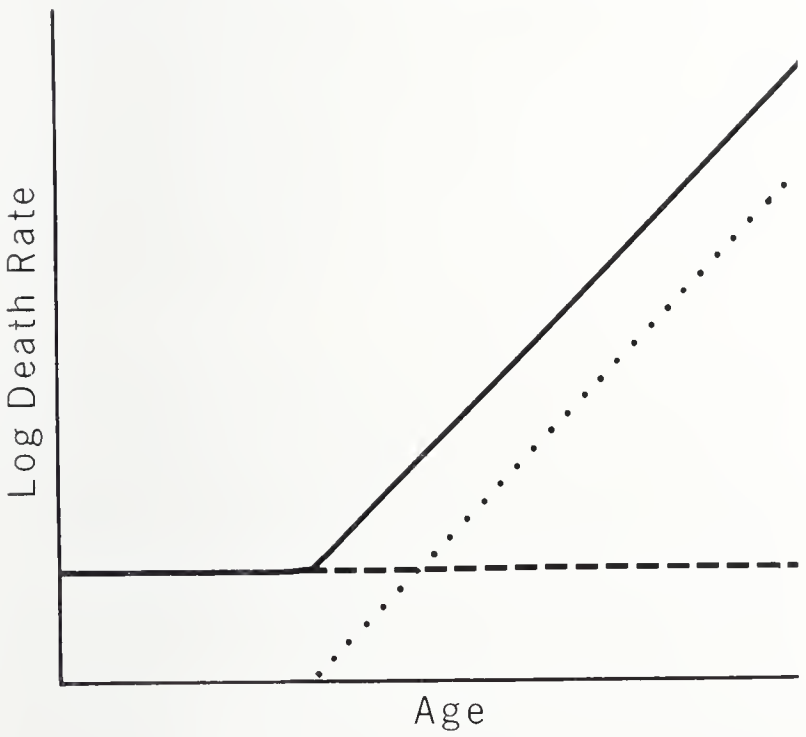
It is only in the last few decades that a concerted effort has been made to study senescence on a systematic scientific basis. Aging processes have been studied in a wide variety of organisms, and although many unanswered questions remain, a few generalizations may be made from the vast assortment of data now available. This paper is a brief survey of some of the current literature that is relevant to an understanding of human aging.

DEFINITION OF AN AGING SYSTEM

It is important to distinguish between a change that frequently accompanies aging and a factor that is inherent to the aging process. To be age-related a change must be a progressive phenomenon that is universal within the population. It must generally be intrinsically related to the organism and not due to its environment.[1] Although some authors consider that development is part of the aging process,[2] most feel that aging consists of deleterious changes and thus begins with maturity,[3, 4] or in the post-reproductive phase.[1] However, not all systems within the organism reach maximum efficiency at the same time, and thus all will not start aging simultaneously.[3]

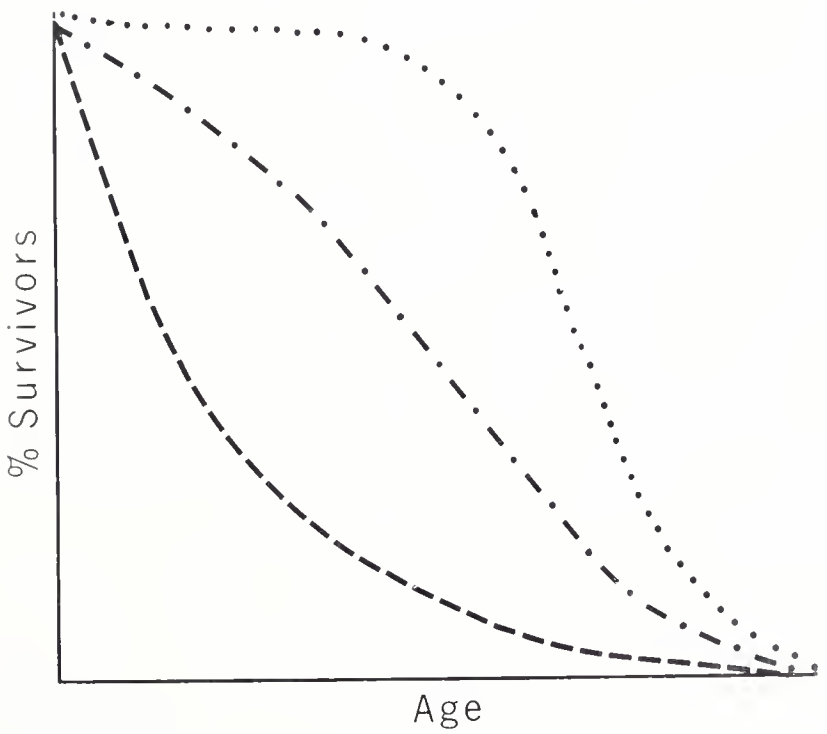
A number of mathematical models have been constructed to provide a rigorous definition of an aging system. The Gompertz plot (Figure 1), derived by Benjamin Gompertz in 1825, is still the basis for modern, more elaborate theories. Gompertz stated that, after maturity, the probability of dying is an exponential function of time.[5] The same data is shown in Figure 2 in which the percentage of survivors is graphed against time. Any deaths that are due to non-age-related disease or accident will tend to move the curve away from that of an age-related system, towards the non-age-related curve.

Figure 1
Gompertz Plot



Redrawn from Kohn (1971)[5]

Figure 2
Comparison of Survival Rates



Redrawn from Kohn (1971)[5]

A dying system may be characterized by two parameters – median and maximum lifespan. If death is due solely to age-related causes, the median lifespan will approach the maximum.[6] Thus, modern medicine has increased man's median lifespan, without affecting his maximum. It is important that this fact be recognized since many modern techniques that claim to increase the length of man's life merely affect the median, and not the maximum lifespan.[6] Even so, the fallacy that changing man's median lifespan will eventually cure aging is almost universally accepted by society.

SENESCENCE IN LIVING SYSTEMS: AN OVERVIEW

A bewildering array of data has been accumulated in recent years describing the effect of aging on a number of species in every major group of organisms. Although much of this may not seem applicable to man, in order to determine the reason for the clear pattern of human senescence it is useful to examine those traits that seem to be associated with a wide variety of senescing organisms, but absent from those with indeterminate lifespans. Therefore, we may be able to determine whether some human characteristics carry the price of "causing" senescence.

Death due solely to senescence is probably a very rare phenomenon in the wild.[7] In most cases aging results in a decreasing ability to cope with stress.[5, 7] Thus, an aged individual will succumb to a level of disease, starvation or increased activity that would readily be handled by a younger member of the species.

An increasing susceptibility to pathogens has been found to be the eventual cause of death in many trees.[8] Arthropods have been observed to die moulting or after periods of intense activity.[7] Older vertebrates, like the very young, are more susceptible to predation. Similarly, aged predators become increasingly unable to capture sufficient food and die from malnutrition, predation or disease.

The same is true of human populations, although to a lesser extent because of artificial protection from natural stresses. Post mortems have shown that a degree of lung damage from pneumonia that would barely be noticed by a young adult will be sufficient to kill an older patient. Senior citizens are also far more susceptible to hypothermia during cold weather and are the main victims of influenza epidemics.

It is difficult to find factors generally associated with aging that apply to all living systems because almost every group of organisms contains some species that have short, clearly defined lifespans, and others that live much longer, if not indefinitely.[7] However, one highly significant generalization has been made by many authors. Those organisms which grow continuously, or otherwise reorganize all their tissues, tend to have extended or indefinite lifespans. Conversely, animals with very defined lifespans seem to have a highly restricted size and growing period, with a limited degree of cell replacement.[1]

This applies to both plants and animals. In fact, unicellular ciliates must undergo sexual conjugation, or another form of complete nuclear reorganization known as autogamy, or endomixus, at regular intervals or the culture will senesce.[9] Among higher plants it has been shown that trees planted by early American pioneers in a climate that prevented flowering have maintained a colony for many centuries by the shooting of their roots, which must have continued to grow long after the original trees had died. Animals such as the molluscs, kept in a colder environment which slows growth, will live longer.[7]

Gravity is an extremely limiting factor for the growth of land organisms.[2, 7] The longest-living land vertebrates are a few large reptiles which grow slowly throughout their lifespan but which have their weight largely or wholly supported by the ground.[7] It is of interest that increased size (and age) may actually decrease the probability of death for a time because of a reduced susceptibility to predation. It is probable that pathogens are the eventual cause of death for the large reptiles, as for the trees. Most other land vertebrates have a definite developmental period and adult size, with clearly defined aging patterns.

Flying animals are even more closely controlled by gravity. Both birds and flying insects tend to be smaller than related groups and are shorter-lived.

Major support for the premise that continuous growth and cellular replacement retards the overall aging process has been provided by experiments with flatworms. If these animals are cut into pieces, each piece will regenerate into a new worm. Although pieces repeatedly taken from the head end tend to be less viable, the other sections will show no signs of aging for as long as the process is continued.[1, 7]

Major tendencies toward specialization appear to reduce the lifespan of the species. The presence of irreplaceable organs or systems, such as the human nervous system and the immune system, limit the lifespan to the extent that the organism is unable to control their degeneration.[10]

A number of organisms exhibit forms of reproductive behaviour that result in death. Many, especially among more primitive forms of life such as certain sponges[7] and some fungi, are physically disrupted by reproduction. Others, like the annual plants, die shortly thereafter. No arthropod is known to have an indeterminate lifespan[7] and, in many, the adult is limited by larval food reserves to the performance of its reproductive function.[2] However, some authors feel that this is not true senescence. Their view is supported by the fact that annual plants, kept alive for several years by preventing flowering, cannot be maintained indefinitely and undergo what might truly be termed senescence.[8]

As usual, mammals have been extensively studied in the laboratory with regard to their patterns of senescence. The length of a species' lifespan has been found to vary directly with its size,[1] the length of its

growth period and degree of cephalization (excess of brain weight over that expected for the size of the mammal) and inversely with its metabolic rate.[7]

Many physiological and cellular changes have been documented in mammals. Nerve conduction velocity and renal blood flow decrease with age.[3] In general, the number of red blood cells decreases,[3] and respiration is less efficient, due to a decreasing tidal volume[3, 11, 12] and decreasing compliance.[11, 12] These factors produce a generalized hypoxia that may be the cause of many cellular changes and of the inadequacy of physiological response to stress.[11]

Attempts to demonstrate generalized changes in hormone secretion (except sex hormone production, which clearly declines)[2] or enzyme production have largely been unsuccessful.[13] An apparent decrease in hormone production is sometimes caused by a decrease in the response of the target organ.[14] Also, autonomic regulation of responses becomes more erratic.[15]

Certain organs such as the brain, kidney[1, 2, 7] and liver[1, 2, 7, 15] decrease in size due to increased cell death[2, 15] or to decreased cytoplasmic volume of the individual cells.[2] Bone resorption exceeds formation, resulting in increasing porosity and fragility.[16] Various other effects of aging will be discussed later in relation to the theoretical models that have been proposed.

THE THEORETICAL BASIS OF THE STUDY OF AGING

1. *Exhaustion of Components*

The theory that aging is due to the exhaustion of certain irreplaceable cellular components formed only during sexual reproduction was first suggested by Butschli in 1882.[7] Although human fibroblasts in culture seem to have a limited proliferative potential that decreases with the increasing age of the donor,[17] there is no evidence that this is limiting during the normal lifespan.[18] In addition, no essential factor has been found that becomes significantly reduced in aged cells, and the theory is now of no more than historical interest.[7]

2. *Accumulation of Toxic Substances*

The only substance that has been found to accumulate in a variety of vertebrates with age is lipofuscin. It is most prevalent in non-dividing tissues such as muscle and nerves.[1, 5, 7] It has also been found in young patients suffering from progeria, which causes premature aging.[19] In addition, mammals which age much faster than human beings accumulate the pigments at a correspondingly increased rate.[2]

The fact that residents of Hiroshima exposed to the atom bomb did not accumulate more pigments than residents of the same area not exposed suggested that lipofuscin formation is not due to mutations within non-dividing cell populations.[1] Centrophenoxine, a drug used to treat symptoms of senility in humans, has been found to decrease the concentration of lipofuscin in the neurons of guinea pigs.[20] However, lipofuscin accumulation is accompanied by the degeneration of cellular membranes, which may be the cause of the symptoms associated with lipofuscin pigments.[19] Lipofuscin may in fact be the result of the peroxidation of membranes by lysosomes, forming a non-metabolizable product.[1]

The significance of the presence of lipofuscin pigments has not yet been determined. However, even if it can be proven to be detrimental it will not explain many of the changes observed in aging mammals.

Although no other substance has been implicated in vertebrates, there is one curious example of the formation of a factor that may cause aging in lower animals. Female rotifers have been found to produce progressively shorter-lived offspring as they age, perhaps due to the production of an aging factor transmitted to their eggs once growth ceases.[4] The effect is reversible, since the short-lived generation will produce offspring when young which have longer lifespans than the parent. Because longer-lived rotifers grow more slowly and reach a greater size at maturity, it may in fact be that younger rotifers transmit a factor that slows the growth of the offspring. No substance that produces either effect has yet been isolated.[1]

3. *The Somatic Mutation Theory*

The observation that the occurrence of chromosomal abnormalities, such as aneuploidy and breakage, directly observable in cells during mitosis, increases with age has led to the suggestion that the accumulation of deleterious mutations causes aging.[6, 18, 20]

Radiation is known to increase the mutation rate, and some workers such as Curtis[21] claim that radiation also accelerates the aging process. However, Alexander[22] states that radiation increases the incidence of cancer but has no effect on the rate of aging. The fact that cancer incidence increases with age in a normal population is propably due to that fact that carcinogenesis requires a series of events of constant probability, including mutation, and the deciding factor in the development of cancer may be an individual's susceptibility to a given mutation. Different types of cancer have sharply defined periods of incidence in the lifespan in many cases,[1] and the overall increase may be a mathematical artifact.[6]

The amount of radiation required to produce signs of premature aging causes a much greater incidence of chromosomal abnormality than is normal in aged cells.[20] However, the level of visible abnormalities decreases with time due to the death during mitosis of many severely damaged cells and to repair mechanisms. The low level of observable aberration in normal aged tissues may not reflect the true incidence of slightly deleterious non-observable mutation.[21] While the loss of post-mitotic cells, particularly of nervous tissue which cannot be compensated by hypertrophy of the remaining cells, may prove limiting, there is no evidence that this is a factor in normal aging.

Paradoxically, small doses of radiation increase the lifespan of some animals,[18] which may possibly be related to the fact that radiation reduces the cross-linkages in collagen (to be discussed later). Chemical mutagens

do not seem to shorten the lifespan unless they are directly toxic.[22]

More decisive evidence against the somatic mutation theory has come from experiments with haploid and polyploid organisms. While radiation (presumably due to recessive mutations) causes a greater shortening of the lifespan of haploid individuals than of diploid and of diploid than of polyploid, under normal conditions haploid, diploid and polyploid individuals have the same lifespan.[23]

4. *Aging of Connective Tissues*

The most studied changes in vertebrate connective tissue have involved collagen, which forms about a third of the total protein in mammals. Collagen is synthesized in the form of tropocollagen molecules, which are soluble in salt solution. As cross-linkages form spontaneously *in vitro* between the molecules, collagen is produced, which becomes increasingly insoluble.

Collagen is not completely inert and forms an important part of the metabolic pool in times of stress.[24] It may be significant that the amount of collagenase in the tissues of rats declines with age, thus reducing its availability as a metabolite when energy requirements are increased.[25] However, much of the body's collagen remains in place for very long periods of time. Gradually it becomes increasingly cross-linked and more insoluble.[5] Age-related changes in the nature or quantity of cross-linkage may make it even less susceptible to replacement. Experiments with rat collagen have shown that cyclic movements, such as occur in the heart and lungs, result in increased cross-linkage and stiffness.[26] Animals with higher metabolic rates (and shorter lifespans) show more rapid cross-linkage.[5] It is of interest that males, who are more vulnerable to cardiovascular disease than females, also have a higher ratio of insoluble to soluble collagen.

The result of increasing cross-linkages is a significant increase in the resistance of lungs, heart and blood vessels to movement.[3] It thus requires greater energy to breathe and to transport oxygen and nutrients to the tissues, producing an inferior physiological environment.[3, 5] Although the organism may be able to maintain itself under normal conditions it becomes increasingly unable to cope with stress. The fact that organs transplanted into younger hosts frequently outlive the maximum lifespan of the species supports the idea that aging is not due to the declining competence of the individual cells, but to their poor environment.[27]

5. *Age Changes in the Immune System*

Two contradictory changes in the immune system have been correlated with age. While there is a general decrease in cell-mediated immunity with age,[3, 5] the incidence of auto-immune disease increases.[3, 6, 28]

The decline in immune response is probably due to a decrease in the number of immunologically competent cells rather than to the impaired function of existing cells. Declining immunity results in an increased susceptibility to infectious disease.[29]

The increase in auto-immune disease is thought to be due to the escape of sequestered cells into the body fluids,[6] or to genetic changes in lymphocytes, which produce unusual antibodies capable of attacking the body's cells.[28] A genetic change might involve a mutation in the genes that produce or control the production of antibodies, or possibly a nongenetic derepression of certain genes. Humoral antibodies have been isolated from the blood of aging mammals which precipitate with antigens on their own kidney glomeruli[29] and neurons.[30]

That auto-immune phenomena may cause premature aging is supported by the fact that progeria is thought to be due to the presence of maternal cells in the offspring. Although the offspring is tolerant to the cells of the mother, the maternal cells produce antibodies that result in the destruction of the tissues of the offspring, drastically reducing its lifespan.[28]

The lack of pathology in many senescent deaths may be due to auto-immune diseases. Certainly, the maximum lifespan of a species of mammal is accompanied by an increase in the incidence of auto-immune phenomena.[28]

GENETIC DETERMINATION OF LIFESPAN

Although the lifespan of an individual is modifiable within species limits, it is controlled by inheritable mechanisms. Female monozygotic twins have a mean lifespan difference of 3.5 years,[31] and there is a significant correlation between the lifespans of parent and child and between dizygotic siblings.[6]

Various factors, such as antioxidants, moderate exercise,[6] disease prevention, and a reduced but balanced diet[2] do increase the mean lifespan of a population, although there is no real evidence that the maximum lifespan can be modified.

The possible mechanism of evolutionary selection of a determinate lifespan poses an increasing problem. Some authors feel that a determinate lifespan resulted inadvertently from the selection of evolutionarily valuable traits not connected with senescence.[1, 7] For instance, the fact that the limitation of size of an organism was advantageous in the face of limited food supplies or gravity, may have resulted in senescence. In addition, specializations, which gave the species a significant advantage but required irreplaceable systems, may have had a similar effect.

On the other hand, there are mechanisms that would permit the active selection of a limited lifespan. The limitation of the reproductive period tends to reduce the chance of lethal mutations in the gonads.[32] Once an organism can no longer reproduce, it is consuming food resources valuable to the rest of the population.[1] Thus a population whose older members were highly vulnerable to increased stress would be more likely to survive periods of starvation or climatic stress than a population whose younger members had to compete

with the post-reproduction members for the available food resources and shelter.

It is obviously impossible to determine the mechanism whereby many species have developed their clearly defined lifespans, and different factors may have operated in each case. However, it does appear that a determinate lifespan is the result of an organism's genetic makeup.

CONCLUSIONS

In the last few years a number of theories as to the causes of aging have been suggested. It is impossible to state categorically reasons for the fact that certain organisms clearly senesce within a defined period of time, whereas others seem to be immune to any aging process. Each factor that is in operation is affected by all the processes that occur within the organism, and it is often impossible to determine if a factor is the cause of senescence or the result of some other process that is the true cause of aging. The controversy over the significance of the accumulation of lipofuscin is an example of this type of problem. In addition, it is difficult to separate the organism from its environment, and any factor that is stated to be the cause of aging must be unmodifiable by any changes in the organism's physical, nutritional or chemical environment.

Aging in most species can be described as a generalized decline in physiological competence, particularly in relation to stress. Individual cells tend to be unaffected by age, except in so far as the quality of their physiological environment is reduced.

At the moment it appears that the major causes of aging in mammals are the stiffening of collagenic connective tissues, with a corresponding decrease in oxygen and nutrient transport to the tissues; the decline in immunity to infection; and the auto-immune destruction of the individual's own tissues. It does not appear that the accumulation of toxic metabolic products is a major factor in aging. Although somatic mutations might be involved in the changes in the immune system, it does not seem that increasing the mutation rate increases the rate of aging.

Our understanding of aging in living systems is in no way complete, and this paper is a very brief survey of part of a vast body of literature on the subject. In the past couple of decades the foundations have been laid that permit scientific investigation of the causes of aging, and a few basic theories have been proposed.

Although, from the evidence that exists today, it appears that the human lifespan is indeed limited, this does not mean that research into the causes of aging is futile. An investigation of the aging process may lead to a greater understanding of normal growth and development, of the interaction between the cell and its physiological environment, and of the factors controlling immunity, and may allow us to control those changes that are found not to be an inherent part of the aging process.

Although we will probably never be able to prevent death, or even extend the human lifespan significantly, the quality of life prior to senescence is of great concern, and the study of senescence may enable every human being to have an active healthy lifespan that approaches the maximum of our species.

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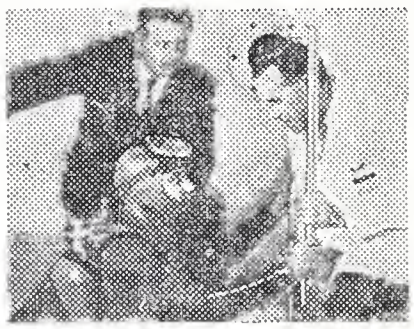

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DRS-70-21

An Interview with a Geriatrician: Dr. Michael Gordon

Frances Frankenburg, 7T8

Could you tell us about your medical training, and how you became interested in geriatrics?

Most importantly, I had lovely grandparents. I went to a medical school called St. Andrews in Dundee, Scotland. I did my first medical internship in Aberdeen and then Israel. I went to Boston in 1967 where I did a year of straight medical internship. Following that, I spent a year as a junior medical resident in Montreal. I returned to Israel, and for six months worked in pathology at the Hadassah Hospital in Jerusalem.

That was the time of the war of attrition with Egypt, and I was persuaded to volunteer for military service. I spent a year and a half in the Israeli air force as a flight surgeon, although I also worked in the neighbouring settlements. I then spent two years in Jerusalem – at Shaare Zedek Hospital. It was built during the Turkish occupation, and the same buildings were being used. It was overcrowded, but medical service was of extremely high quality and unusually varied. I was senior resident and chief resident, and ran a geriatric service – a new idea in Jerusalem – for eight months. This proved to be an important period of my medical training.

I returned to Canada, and after a year of nuclear medicine at TGH, accepted the position of chief resident in internal medicine at Mount Sinai Hospital. During this time I made my contacts with Baycrest Centre and following my residency I started work there as a consultant in internal medicine with a special interest in geriatrics.

What does your work consist of now at Baycrest and Sinai?

At Baycrest Hospital I supervise the interns, am available for consultations concerning general problems, and mediate between the hospital, the attending physicians, and the subspecialists at Mount Sinai Hospital.

In addition, I am involved in plans for the Baycrest Terrace – a facility for the well-elderly. It will be for people who may not need medical support as much as they need social supports. They will be quite independent – for example, they will be served only one meal a day. It's a novel and exciting concept. Hopefully it will fill an important need in the community.

At the Mount Sinai, I teach and I practice general internal medicine, with a special interest in geriatric care. I see elderly patients with medical problems and and those who may be candidates for care at Baycrest Centre.

My work at Sinai complements my work at Baycrest nicely – I enjoy the teaching, and by seeing nongeriatric patients I do gain wider experience which I take back to my work at Baycrest.

Returning to the question of your training – is there any special training in Canada for those doctors interested in geriatrics?

There is no specialty recognized in Canada as geriatrics. If you decide to take care of old people, then you're a geriatrician.

Is there a need for special training?

Yes, since the elderly do have special problems. For example, they handle drugs in a way quite different from younger people. Whether this is due to abnormalities of liver function, body mass, distribution, absorption or excretion is not clear. Adverse reactions are more of a problem for the elderly. They often have many diseases simultaneously for which they are being treated, and the problem of drug interactions can be a serious one.

The integration of all their problems and an approach to the total patient is very important. The older person does not adapt to change very well – having to move from one hospital or facility to another may disrupt his equilibrium. At Baycrest we try to integrate his needs. For example, we use the problem-oriented charting system, so that all members of the health care team – doctors, social workers, nurses, therapists, podiatrists – are aware of all the patient's problems and can all contribute to his total care.

Many people have difficulty in understanding how a practice made up of old people could be satisfying. Could you comment on this?

It may be easier to care for a young person whom you can see get better. However, if you look at most of the Toronto hospitals, you'll see that they're often treating geriatric patients. Of course, in a geriatric centre there is a narrower spread of ages – from elderly to ancient.

Not all of the people I see are sick. The people applying to the Baycrest Terrace, for example, are in their 70's, 80's and 90's, and do not want their activities or independence limited in any way. They may have had a recent medical problem which has frightened them somewhat and they now want some support. They do not want or need institutionalization. These people are not sick, and certainly do *not* think of themselves as old.

Among elderly people you can see the spectrum of medical diseases and human qualities. By talking to many of them, one can hear things first hand that most people can only read about. They can be fascinating, and often have a great deal of wisdom. The job satisfaction is no less than I would expect in a general medical practice.

Would you describe the Baycrest Hospital?

The hospital is one part of the Baycrest Centre. The Centre also contains the Jewish Home for the Aged, a day care program, and the soon to be opened Terrace for the independent elderly. The Centre is absolutely committed to the wellbeing of its population. There is also a group of extraordinary, dedicated volunteers who work with the patients, serve on the advisory board, and assist with financial support. I do most of my work at the Hospital.

Who are the patients in the hospital?

A third of the patients are there for terminal care. They are a responsibility of the medical profession, and unfortunately many health care workers would prefer that someone else assume that responsibility. The Hospital does take care of many people who are dying, and takes care of them well. We give them and their families as much support as possible. We are in close contact with the oncologists at Mount Sinai and Princess Margaret Hospitals, and we occasionally are able to restore some independence to people who have been admitted for terminal care.

Another third are admitted for long term chronic care. Somebody must look after these people. As much as we can, we maintain the quality of their lives.

The final third are there for rehabilitation – for example, they will come to Baycrest after suffering a fracture, a stroke, or a severe medical illness. They generally go home or on to another facility such as a nursing home.

How is the break-up of the nuclear family affecting geriatric practice? Do you see this as a problem peculiar to our Western society?

I remember being surprised in Israel to see the family unit breaking up because I had thought that there, especially with its large Eastern population, the family would still be very strong. But, for various reasons, it is disintegrating at a rate probably equal to that here.

The older person today is often isolated physically, emotionally and socially from his family. In return for what their parents did for them, people now do for their own children. We now have a society in which everyone looks to youth.

The community has taken on and will have to continue to take on responsibility for the elderly, without taking away their dignity and independence. Such simple things as designing convenient public transport systems have to be considered. There are myriads of similar changes that could make life much easier for our elderly.

I don't think that we're propagating a sufficient sense of respect or concern for the elderly. Our society has chosen to ignore the fact that there is only a temporal difference between youth and age – and that just as our elders were yesterday's youth, we are tomorrow's elderly.

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Education in Geriatrics

*R. C. Laird, M.D.**

Geriatrics is defined as the medical care of the aging population. With the improvement in general medical care, the percentage of this aging portion of the population is increasing. Geriatrics is a very broad field, encompassing all specialties including that of family practice. As the group of aging people increases in size, our interest in their health must also increase. Disease processes are the same for all ages, but the reaction to these processes must be different when other factors are involved. The aging process in humans has been studied for years by biologists, and many facts have been established, but there are still many unanswered questions. Clinically we know that older people require more careful investigation, are more prone to shock, and are less resilient in their reactions. We feel that these and other differences should be brought to the attention of medical students as undergraduates, interns and residents, and practising physicians, whose practice in many instances consists of almost 50 per cent old people.

For undergraduate teaching it is important to introduce the subject early in the course, and continue throughout, with more time as clinical experience increases. As there is a large social and psychological factor in the approach to older people, there should be, in first year, a lecture or two by the Department of Behavioural Science. This should be accompanied by discussion of the biology of aging, and a presentation of the complexities and extent of the practice of geriatrics. In second and third years, with the introduction of system teaching, there should be a presentation in each topic of the application of the subject to the health of the aging population. There are already available specialists in almost every field who have a definite interest in geriatrics and advantage must be taken of this interest.

The establishment of geriatric units should be mentioned. Obviously this is not essential to the good care of the elderly, but there are certain advantages that should be noted. From the nursing point of view, it has been found that with experience on a geriatric ward, the nurses develop a sympathetic dedication to the old people which is a tremendous help in their recovery. From the medical point of view, history taking, physical examination, and special investigations are geared to the abilities of the elderly, and the effects of the aging processes are recognized and allowed for. From the teaching point of view, both undergraduate and graduate, the segregation of geriatric problems and their treatment focuses the interest of the student and eventually increases his knowledge, but also teaches him the disposal of the patient and the facilities available.

*Provincial Geriatric Study Centre

In the final year of the medical course, now mostly organized as a clinical clerkship, the student should have a definite period of exposure to geriatrics, either in medical or in family practice. The subject could also take its place among the electives, where the student could have six to eight weeks' exposure to the problems.

The introduction of residencies in geriatrics has been very slow in Canada and the United States, but there are now several areas in Canada where this exposure is available. Most of the positions, so far, are in the regular postgraduate training in internal medicine, but a tendency is now developing to include this in the family practice training. The actual organization is not so important as the exposure of the postgraduate students to the problems of geriatrics, and to the knowledge of the facilities available for the care of the aged in any community.

The third area of teaching of geriatrics is the later postgraduate period. For many years the Kellogg Foundation supported this type of education in Ontario, arranged through local medical societies. This is continuing without the Foundation support, and it is an ideal instrument for spreading an interest in the problems of geriatrics. I feel very strongly that the Ontario Medical Association should co-operate with the medical schools of Ontario in expanding the dispersal of information with regard to the problems of the aging population, to the family practitioners of the province.

Let me summarize the facilities available for sick old people at present, or easily available if the need is accepted. The general hospital is essential in the treatment of acute disease, and in-patients may be followed in out-patient departments following discharge. For short-term extended care there may be the convalescent hospital. For longer extended care, there is the chronic hospital. Both of these could very efficiently be part of a general hospital organization, but this has not been generally accepted. Home care organizations have been very satisfactory, and have been successful in reducing the demand for beds in the facilities mentioned. An extension of this which is particularly applicable to senior citizens is the Day Care Centre. This is something like an Out-Patient Department, but it makes available to the patient physio- and occupational therapy, as well as some nursing and medical care. This facility is not yet commonly available here, but is a development that should be encouraged. Both nursing homes and Homes for the Aged are providing extended care, as well as domiciliary care. Most of these facilities are available now for undergraduate and postgraduate teaching of medical students and doctors, and they should all be used if we hope to give a true picture of the health of the community.

Marilyn R. Gruneir, B.A.

The Role of a "Patient" in a Hospital Setting

Our society has traditionally emphasized the Protestant work ethic coupled with the need to acquire independence. However, when a person becomes ill and is admitted to a hospital he assumes a new role, that of "patient". This role frees him from his daily activities and makes him dependent upon others for his wellbeing. His independence, endorsed by society as a prerequisite for maturity and a very desirable characteristic, suddenly becomes a negative trait. The patient's self-sufficiency is also regarded as undesirable and he is expected to relinquish both immediately upon admittance. These traits have been built up over many years and yet, upon admission, the patient is told to "disregard what you have been striving for all your life. In this situation you are no longer an adult, you become a child once again. You will be told what to do and when to do it, you will be cared for – you need only to submit."[1]

The effects of hospitalization will of course vary with the individual and his or her particular medical and emotional needs. A hospital places everyone in a protective, sterile environment where they are freed from both the responsibilities and stimulations of their homes. Surroundings are alien and unfamiliar, and new and unusual routines and restrictions are imposed by the staff and the environment. In addition, patients find that they are no longer master of their own destinies. They are forced now to give up control over their simplest everyday functions – from the time that they awaken, to what and when they will eat. These and many other details of everyday living are no longer determined by their own likes, dislikes and habits, and they are expected to submit without question.

"The need to give up the right to make decisions both as to the details of everyday living and as to what is to be done to him, the physical demands of the illness, the feeling of helplessness, and the necessity to rely upon others for the satisfaction both of physical and of emotional requirements, all tend to centre the patient's attention upon himself and to make him a completely dependent individual."[1] As long as they remain in hospital total dependency is enforced, the effects of which are often lost sight of in the busy day to day workings of the hospital. The patient is left on his own to cope with this when he returns home.

Patients vary in their ability to relinquish the dependence which hospitalization supports. For those who have achieved independence at great cost and after a long struggle, the delicate balance they have managed to maintain may be seriously threatened by a new conflict between dependence and independence. "For those patients who feel inadequate in the face of the competition and trials of the workaday world, discharge may represent a threat; for it means that they are once again being brought face to face with the problems from which hospitalization removed them. No matter how unsatisfactory the hospital experience may have been, the world outside may be even more threatening."[1] It

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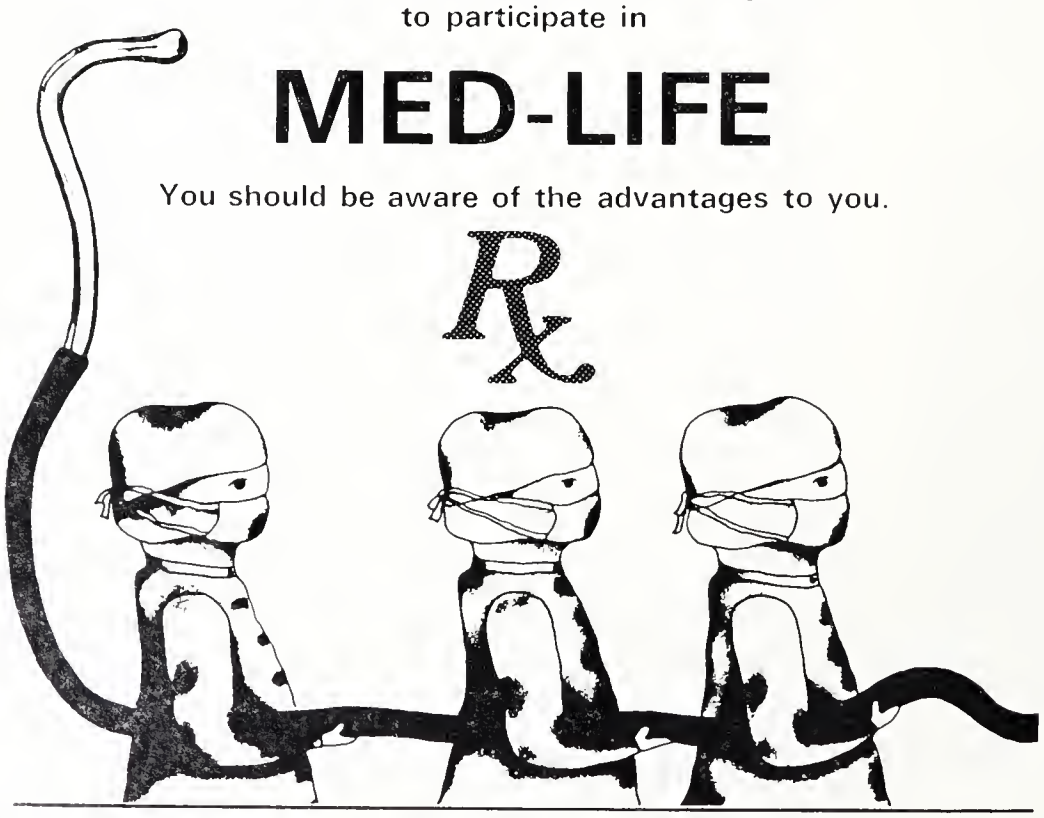

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is not surprising that many people react to discharge with extreme anxiety, fear and resistance.

Regardless of the patient's response to discharge, the fact remains that he must make an adjustment, one which is often difficult. A social worker can work with the patient to make the best possible discharge arrangements but he cannot transform the hospital experience. It is extremely difficult to plan for a person's return to his former independence when he has become accustomed to the passive life of a hospital.

Treatments, clinical appointments and therapy schedules must be prearranged to accommodate the large number of hospital patients. Aside from these and other life-sustaining procedures, individual choice should be encouraged wherever possible. The medical team has a very demanding job, and I am suggesting they change not their work pattern but rather their approach. Instead of a nurse taking a dress from the cupboard and handing it to a patient, no more effort would be involved in giving the patient her own choice of dresses. By keeping the patient involved in his own decision-making each day his independence will not be totally lost.

As the patient's dependence increases his individuality decreases and sometimes disappears. Thought must be given to the long term effects on the patient and the repercussions which might occur when he is discharged. What has become of human dignity and human rights? Are hospitals treating "patients", or people who are individuals with the right to be their own masters?

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Soft Tissue Injuries of the Knee ARE Important

CASE PRESENTATION

Mrs. M.W., a 32 year old teacher with no previous history of knee injury, was first seen in the office on the twelfth of January 1976. She complained of an extremely painful and swollen right knee of nine days duration, associated with much difficulty in weight-bearing.

She stated that while skiing in Quebec she had hurt her knee and fallen forward, and her bindings had failed to release. She was able to make it down the hill unassisted. At that time the knee was extremely swollen and painful. As the symptoms persisted, the patient went to the emergency department of the local hospital. Plain films of the joint revealed no injury and the patient was sent home with instructions to wear a tensor bandage and to use crutches, and with the assurance that the injury sustained was inconsequential. Nine days later, as the knee continued to give the patient much pain, she consulted an orthopaedic surgeon.

On examination the right knee was diffusely swollen, measuring 38 cm in diameter, compared to the 29 cm of the left knee. An effusion of approximately 75 cc was present. There was marked tenderness over the tibial insertion of the medial collateral ligament. The anterior drawer sign was positive. The knee joint opened medially and was grossly unstable. The clinical impression was rupture of the medial collateral and anterior cruciate ligaments, with possible damage to the medial meniscus. Stress x-rays and arthrograms were deferred as the clinical diagnosis indicated the need for surgery.

On January 15, 1976 Mrs. M.W. underwent surgery for the repair of her anterior cruciate ligament by a Modified Jones Procedure, reattachment of the peripheral edge of the medial meniscus to the medial collateral ligament and reattachment of the medial collateral ligament to the tibia. The limb was immobilized in plaster for six weeks, flexed ninety degrees in neutral rotation. The cast was then removed and stability of the joint was assessed. The patient was given two weeks to mobilize on her own. Eight weeks postoperatively the patient was seen again to assess range of movement of the joint. The operative incisions were well healed but the quadriceps was found to be wasted, especially the vastus medialis. Slight movement of the tibia on the femur and minimal medial instability were detected but were inconsequential in terms of function. The patient was referred to physiotherapy.

DISCUSSION

The injury complex described above involving damage to the anterior cruciate, medial collateral ligaligament and medial meniscus has been named *O'Donoghue's Triad*. Before an analysis of the mechanism of such an injury is discussed a review of the of the anatomy and function of the soft tissues of the knee is in order (see Fig 1).

The intrinsic instability of the bony structure of the knee is readily apparent when one visualizes the femoral condyles rocking anteriorly and posteriorly on the flat tibial surface. The stability of the joint is provided by the soft tissues which consist of the internal and external ligaments and the semi-lunar cartilages. Damage to the soft tissues results in instability of the joint and interferes with its function.

The anterior and posterior cruciate ligaments are the important internal ligaments. The anterior cruciate runs from its postero-lateral origin on the medial aspect of the lateral femoral condyle through the intercondylar groove to its insertion antero-medially on the proximal tibia. This ligament provides medial and lateral rotatory stability and resists anterior motion of the tibia on the

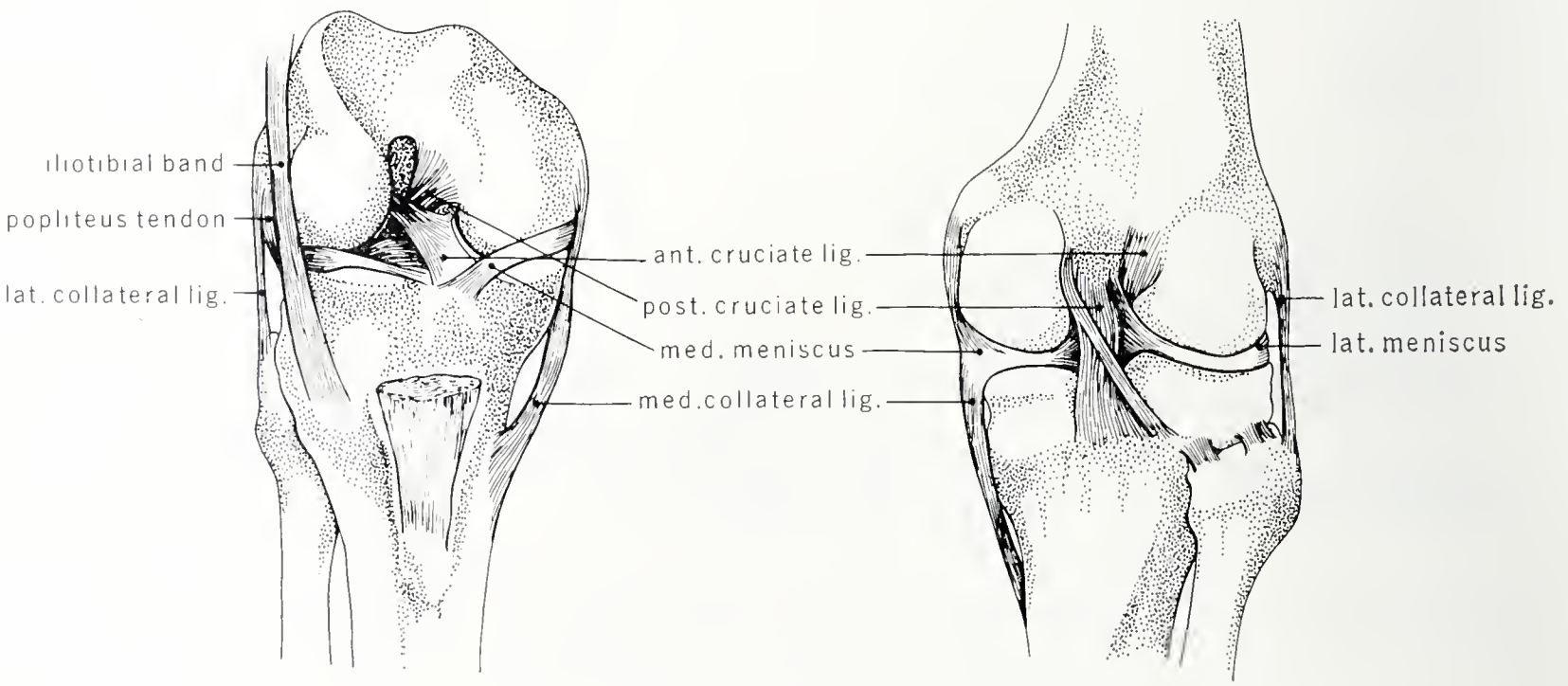


FIGURE 1. Normal Anatomy of the Knee

femur. The posterior cruciate courses postero-laterally from its antero-medial origin on the lateral aspect of the medial femoral condyle to its tibial attachment. The posterior cruciate serves to limit posterior movement of the tibia on the femur, as well as to prevent hyperextension.

The external ligaments form a sleeve about the joint. Medially, stability is maintained by the medial collateral ligament, with its long fibres arising on the femoral condyle and inserting below the pes anserinus. Shorter capsular fibres arise with the long fibres, but insert more proximally on the tibia. The deep capsular fibres engage the outer rim of the medial meniscus. Laterally, the lateral collateral ligament along with the popliteus tendon and fibres of the ilio-tibial tract function in resisting varus stress to the knee. In conjunction with the medial collateral, the lateral collateral provides some rotatory stability and limitation to antero-posterior movement of the tibia on the femur. Anterior stability is maintained by the patellar tendon and retinaculum, as well as by the anterior cruciate. The posterior capsule resists hyperextension, assisted by the tendons of semimembranosus, semitendinosus and biceps femoris, and the posterior cruciate. When the knee is extended, the posterior capsule provides some degree of medial and lateral stability.

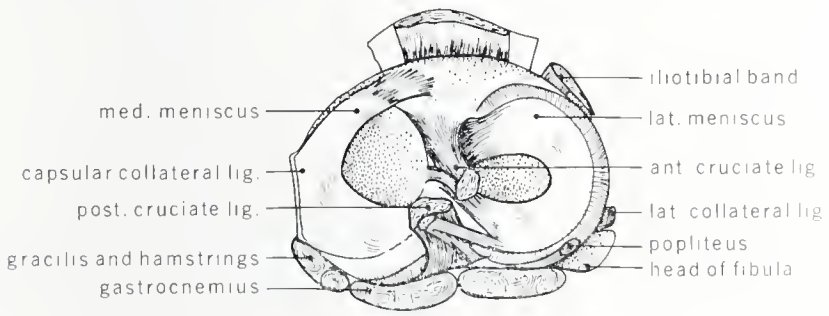


FIGURE 2

The menisci are basically crescent-shaped pieces of fibrocartilage (see Fig 2). Both medial and lateral menisci attach to the cruciates posteriorly, while only the lateral is bound to the anterior cruciate by the superficial transverse ligament. The medial semilunar cartilage is attached to the medial capsular ligament and is frequently involved in injury to the medial collateral. The menisci move with the tibia and allow smooth motion of the tibia on the femur. These cartilages also function to dissipate direct stress through the joint, and provide some rotatory stability.[2]

Understanding the structure and function of the knee permits one to propose the *mechanism producing the injuries of O'Donoghue's Triad.* As the ligaments of the knee act in conjunction to produce stability, it is reasonable to anticipate damage to more than one ligament in a valgus stress injury. In a study by R. L. England,[1] 53 per cent of major ligamentous injuries involved both anterior cruciate and medial collateral ligaments. O'Donoghue[2] feels that involvement of the medial collateral, anterior cruciate and medial meniscus occurs when a valgus stress is placed on the flexed knee, while the tibia is held in external rotation. Nicholas[3] supports this view and carries it further suggesting that the antero-medial corner of the tibia moves forward and twists laterally with respect to its normal relationship with the femoral condyle. Such a force results in strain and rupture of the medial collateral ligament and possible detachment from the medial meniscus posteriorly. In this position the anterior cruciate is stressed and may rupture as it passes over the lateral condyle of the femur. If the knee is extended rather than flexed, it is likely that the posterior capsule and possibly the posterior cruciate will also sustain injury. It would therefore be reasonable to assume that Mrs. M.W. received her injuries when her ski boot, failing to release from the ski, held her tibia in external rotation while she catapulted forward, producing a resultant valgus stress on the knee.

The importance of early diagnosis and treatment of severe ligamentous injury cannot be overemphasized. Failure to correct the damage may result in joint instability, predisposing to further injury and osteoarthritis. In a study by E. H. Simmons[4] of Toronto involving 189 patients who had undergone repairs of the medial collateral or anterior cruciate, or combination thereof, stability of the joint was good in 70 per cent of patients undergoing surgery within ten days of injury, while only 50 per cent of those having surgery two weeks to three months from the time of injury achieved comparable results.

Assessment of knee injury is relatively simple if one follows certain guidelines. An accurate history is essential, with special note taken as to whether or not swelling occurred immediately or was delayed twelve to twenty-four hours. Immediate swelling is consistent with serious injury. The direction of forces causing the injury may be helpful. Whether or not the patient could walk on the injured limb is also significant. On examination, tenderness over the sites of injury is a common finding. Manipulation is routinely carried out on the good limb to reassure the anxious patient and to establish the normal range of motion. This is most important in the younger patient where some degree of antero-posterior movement of the tibia on the femur as well as medial and lateral laxity is normal.[3] The patient is requested to put his knee joint through its range of motion. Abnormal mobility and positions associated with pain are noted. Tests of flexion-rotation with the knee in internal, external and neutral rotation will help establish the presence or absence of meniscal damage, as well as demonstrate rotatory instability. The finding of anterior or posterior mobility of the tibia on the femur (posterior drawer sign) is consistent with meniscal damage. To test the integrity of the posterior capsule, valgus and varus stresses may be applied to the extended knee or an attempt to hyperextend the knee may be made. Finally, with the knee flexed twenty degrees, valgus and varus stresses are again put on the joint. In this position the lax posterior capsule is unable to exert any medial or lateral stability and damage to the collateral ligaments is unmasked. These last tests are extremely painful and are generally done under general anaesthesia.[2] However, if done very soon after injury, anaesthetic may not be required as the knee is not exquisitely tender at this point.

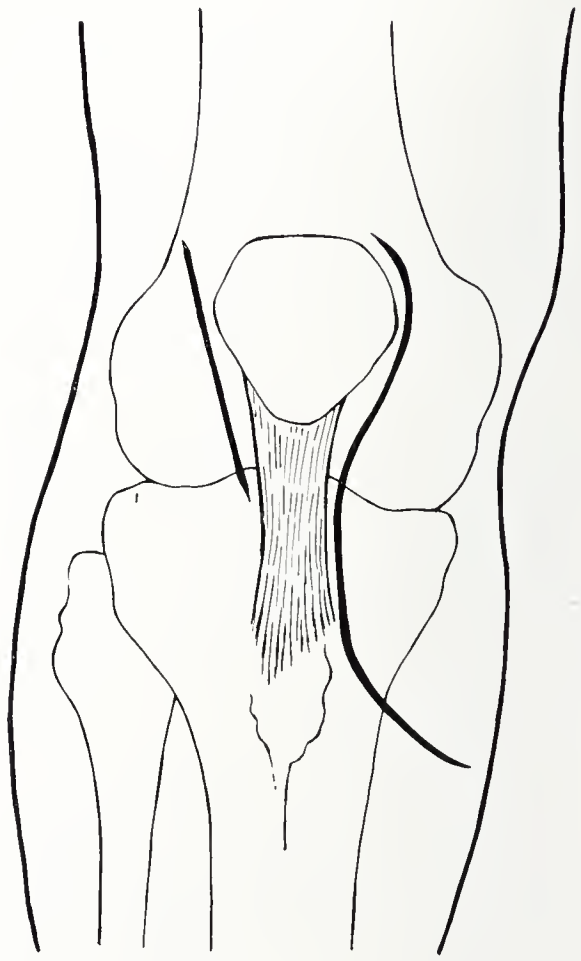
X-rays may be important diagnostically, if taken with the goal of detection of soft tissue injury as well as

fractures. Stress x-rays are indicated when there is a strong index of suspicion of ligamentous damage without clinical confirmation of such and a fracture has been ruled out by plain films. As valgus and varus stressing of the joint with ligamentous injury causes tremendous discomfort, these radiographs are usually done under general anaesthesia or Demerol analgesia.[1] Plain films of the joint without standard positioning are sometimes more helpful than standard views, as positioning the limb may realign the bony deformity. Arthrograms are useful in the delineation of meniscal tears and rupture of the joint capsule, but are often omitted when surgery has been indicated on other grounds.

Injuries to the knee may be classified as mild or severe. With a mild sprain the patient will complain of some tenderness and swelling, but joint function will be unimpaired. Treatment is symptomatic consisting of rest and cold compresses to reduce the swelling. Later, hot compresses may be used to inhibit inflammation. Severe injury involves rupture of an entire ligament, in contrast to the mild injury in which only a few of the fibres are torn. The severe injury is associated with extensive disability and pain. Haemarthroses are almost invariably present. Clinically there is abnormal joint mobility. On occasion, stress x-rays must be resorted to in order to confirm the degree of injury. Surgical repair is the treatment of choice in severe injuries. If surgery is contraindicated, as in the severely debilitated patient, treatment consists of immobilization of the limb in neutral rotation and ninety degrees of flexion for six weeks.

The choice of *reconstructive surgical procedure* to be carried out depends upon the extent of the damage, the preoperative condition of the joint and the age and general health of the patient, as well as the surgeon's experience with different reconstructive techniques. One must also take into account the importance of a stable knee to the patient. In this case it was felt that Mrs. M.W., being young and active, required an attempt to restore complete function and stability.

Several methods have been devised for the reconstruction of the anterior cruciate ligament. The Hey Groves technique, first described in 1920,[5] involves severing of the fascia lata from its tibial insertion and redirecting it through a drill hole in the lateral femoral condyle, antero-medially across the joint space and through a second drill hole in the tibia. The fascial strip is the anchored antero-medially on the tibia. This technique was used until the early sixties when it was superseded by grafts producing greater strength. The Campbell method, introduced in 1939,[6] utilizes the medial patellar retinaculum and tendon. This band of tissue is threaded through drill holes as in the Hey Groves operation, but instead terminates rather than originates on the femoral condyle. The problem with this operation is that the weakest portion of the graft, the patellar retinaculum, traverses the joint space, and is consequently under the greatest stress. In 1963, Jones[7] published his technique of anterior cruciate reconstruction from the medial one third of the patellar tendon. The tendon graft is passed through drill holes in the tibia and femur, and is fastened over the lateral femoral condyle. This technique was largely ineffective as the graft had insufficient length to allow proximal attachment.



Parapatellar and lateral condylar incision used in the Modified Jones Technique

FIGURE 3

In the case, reconstruction of the anterior cruciate was by the Modified Jones Procedure. A medial parapatellar incision was made as in Figure 3. The central one third of the patellar tendon was divided and, together with a shaving and fascial strip from the middle of the patella and midportion of the quadriceps tendon, was stripped down to the tibial tuberosity where the graft was slipped through a tibial drill hole, as illustrated in Figure 4. The gap in the patellar tendon is then sutured closed. A trough was created at the original origin of the anterior cruciate on the femur and the tendon was then passed through the condylar notch and firmly secured above and behind the lateral femoral condyle through a lateral incision over the condyle. The graft is strong and passes through the joint in approximately the same position as did the anterior cruciate. The results of this technique are encouraging. In a study by Alm[8] 66 per cent of patients with this reconstruction were subsequently as active in sports as they had been prior to injury. A further 30 per cent had significant improvement, and had no disability in daily activities or light athletics. Failure to improve in 4 per cent was judged due to osteoarthritic changes in the joint. According to Eriksson[9] in a recent study, the

Modified Jones Proceedure resulted in stability of the joint in 80 per cent of patients one year postoperatively.

Repair to the medial collateral ligament varies according to the degree and location of the injury. When the ligament is simply torn from its bony attachments it may, in most cases, be reattached to the bone by barbed staples or sutures. Rupture in the midportion of the ligament may be corrected by the use of several heavy interrupted sutures. Several complex methods of re-establishing medial stability in more chronic cases have recently been described by Nicholas,[10] O'Donoghue[11] and Hughston.[12] As the medial meniscus had simply been torn from its attachment, menisectomy was not indicated, as this proceedure is used only when the meniscus itself is torn. Repair to the medial aspect of the knee was achieved in this case by resuturing the medial meniscus to the capsular ligament and stapling the torn medial collateral fibres back to their tibial insertion.

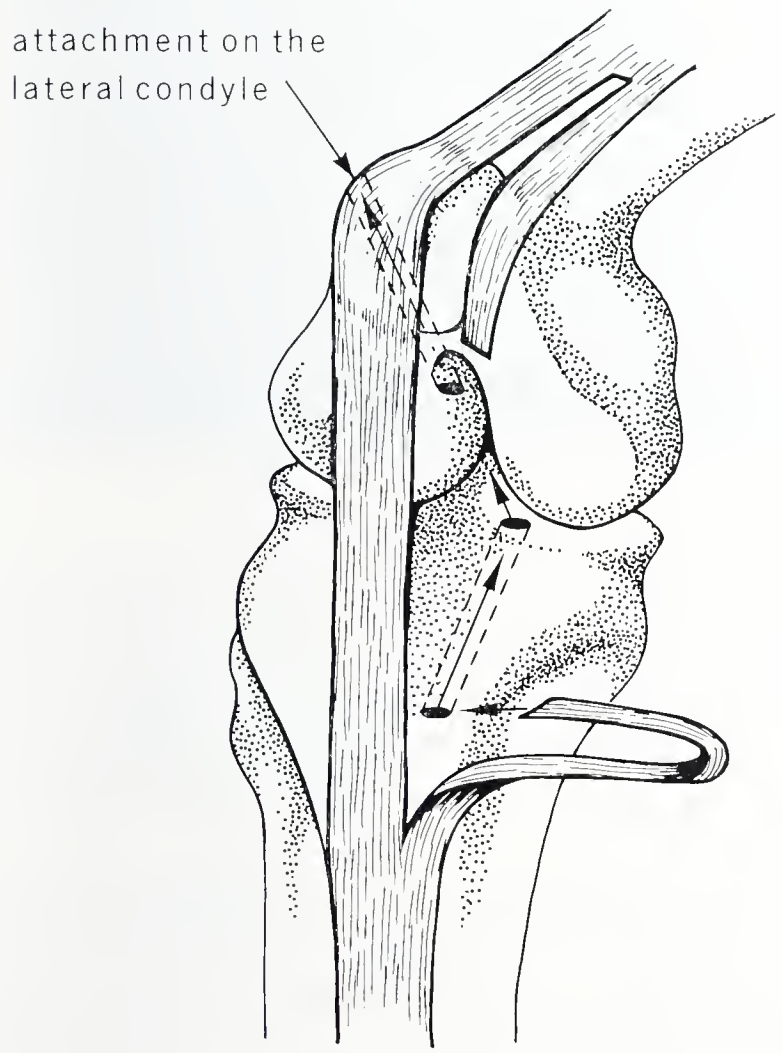


FIGURE 4

CONCLUSION

Although it is impossible to establish in retrospect the initial degree of Mrs. M.W's injury, one would suspect that she had sustained a severe sprain. Further investigation and more vigorous treatment of the injury when she first presented were warranted.

Had the diagnosis never been established, and had there been no surgical repair, the medial collateral ligament would have healed by secondary intent with the production of an excess amount of scar tissue resulting in laxity of the ligament. The anterior cruciate would not have healed and this, in conjunction with the lax medial collateral ligament, would have given rise to antero-medial instability. This instability can lead to further injury, or severe curtailment of the patient's activity. The patient may then require late reconstructive surgery which would be more extensive, and is often associated with inferior results. Uncorrected, the damage the patient sustained would predispose her to osteoarthritis. However, as Mrs. M.W. received early surgical treatment, she has an excellent chance to regain complete stability and function of her knee joint.

This case clearly illustrates the need for early diagnosis and adequate treatment of soft tissue injuries.

ACKNOWLEDGMENTS

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Grow old along with me!
The best is yet to be,
The last of life, for which the first was made
Our times are in his hand
Who saith, "A whole I planned,
Youth shows but half; trust God: see all,
nor be afraid!"

ROBERT BROWNING

Injuries to the Ankle Joint

Kenneth R. Melvin, 7T7

Ankle injuries are among the most common injuries seen by emergency departments. They are important in that lack of treatment may lead to decreased function, increased susceptibility to injury and degenerative joint disease. The following case illustrates the value of a thorough knowledge of the anatomy of a joint and simple mechanical principles in order to deduce both the nature of the injury and its proper treatment.

A 29 year old male presented to the emergency department of a downtown hospital on August 10, 1975 complaining of a painful swollen right ankle following an injury in a football game. The patient stated that while running on a grass field he went over on his ankle. His foot twisted inward under him (thus describing an inversion injury to the ankle), and he felt an immediate pain on the lateral side of his ankle causing him to "see stars" and to feel slightly nauseous. He was able to put little weight on it and was helped off the field. He was brought immediately to the hospital.

On examination the patient was in mild distress due to the pain but otherwise looked well. Examination of his foot showed an area of swelling over the lateral aspect of the ankle which was painful to the touch. There was marked local pain over the distal third of the fibula with some mild reddish-purple discolouration; the patient refused to bear weight. There was also diffuse pain and swelling on the medial side. Sensation was normal over the foot and both dorsalis pedis and posterior tibial arteries were palpable.

FIGURE 1
Anteroposterior x-ray of right ankle

FIGURE 2
Lateral x-ray of right ankle

The patient was x-rayed in the AP and lateral position (Figures 1 and 2). It was noted that there was an undisplaced spiral fracture through the shaft of the fibula. There appeared to be no other injury. On the basis of the history it was decided to put the patient's leg into a below-knee cast in the position of eversion in order to rest the lateral ligaments of the ankle and immobilize the fracture site.

At this point the orthopaedic resident appeared and having listened to the patient's story and viewing the x-rays, noted the following incongruity between the two. Although he maintains that he inverted his ankle when he fell, the x-ray picture could not be produced in this way.

Consider the anatomy of the bone and soft tissues. The tibia and fibula are anchored to the talus and calcaneus by a number of strong ligaments. On the medial side, the deltoid ligament originates on the distal malleolus and courses downward anteriorly and posteriorly to insert in the talus, calcaneus and navicular bones.

On the lateral side, the fibular ligaments pass as the calcaneo-fibular ligament down and posteriorly and the talocalcaneal and anterior talofibular ligament inferolaterally. Between the tibia and fibula both

anteriorly and posteriorly there are the anterior tibiofibular ligaments and the posterior tibofibular ligament just superior to the talus. A few centimetres higher is the interosseous ligament which runs between the tibia and fibula.

These are the important ligaments of the ankle involved in sprains, ligament tears, and fractures. The physiological movement of the ankle occurs at three places through three joints:

1) Tibiotalar– the true 'ankle joint'
2) Subtalar joint } the 'hind foot'
3) Midtarsal }

It is movement at the subtalar and midtarsal joints that primarily cause the inversion and eversion injuries. With fractures, a degree of rotational force, usually external, is applied at the tibiotalar joint.

It is useful to consider the injuries to the ankle in terms of inversion and eversion of the hind foot.

INVERSION

In an inversion injury of the ankle, the stress occurs on the lateral ligaments of the fibula. The inversion causes the ligaments to pull *downward* in a fashion similar to the pulley diagram seen in Figure 3.

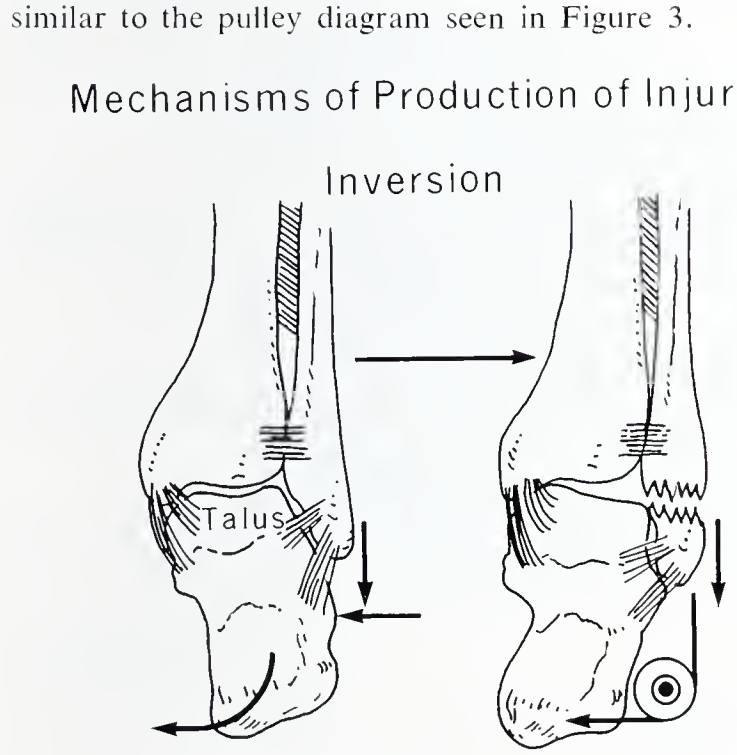

FIGURE 3

It would seem that the insertion of the ligaments on the talus and calcaneus pull the ligaments downward as the bone moves in a downward and internal rotation movement, having the effect of a 90 degree pull at the origin of the ligament, ie, a direct downward pull on the malleolus. The results may be one of three possibilities:

1) the ligaments may stretch, producing pain and no tear (partial);
2) the ligaments may tear (complete);
3) the ligaments may avulse a portion of bone from the fibula.

Note that the force on the fibula is approximately 90 degrees and can only pull off a fragment by a direct pull. Hence, this fragment is going to show a transverse or oblique line most likely below the fibular ligaments.

Injury to the medial side of the ankle is unusual.

EVERSION

On the other hand, eversion injury produces a much different picture. Here the stress occurs on the medial ligament originating on the tibial malleolus.

The eversion causes the ligament to pull downwards on the malleolus at approximately 90 degrees similar to the pulley diagram seen in Figure 4. In this case it is again possible for the deltoid ligament to:

1) strain
2) tear
3) avulse a portion of the medial malleolus in a manner similar to that described for the eversion injury. Again the avulsed piece will show a transverse or oblique fracture line (ie, at right angles to the line of pull).

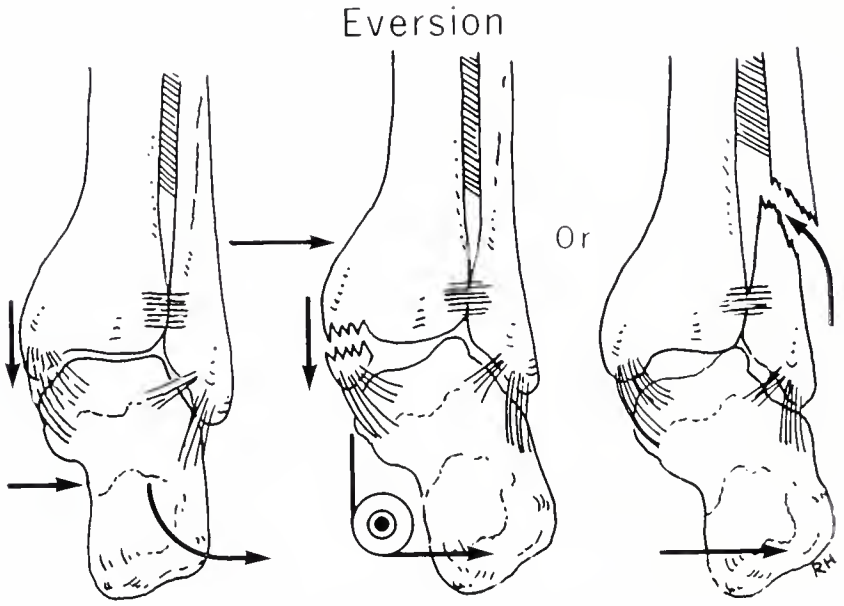

FIGURE 4

However, also to be noted here is the fact that the eversion force may also be transmitted up to the fibula which may break causing a fracture below the joint line or rarely above the tibiofibular ligament. This fracture is a displaced spiral break.

The rupture of the inferior tibiofibular ligaments is known as tibiofibular diastasis. It may be detected on x-ray by observing an increase in the joint space between the medial malleolus and talus, but should be suspected with any ankle injury producing a high fibular fracture. It is caused by lateral shift of the talus and implies that the deltoid ligament has torn or avulsed a portion of the medial malleolus.

It is usual for the injuries to occur in specific patterns and not as isolated events. Thus it is common to see the inversion of the ankle produce trauma to the lateral

ligaments originating on the fibula. Severe or violent injury may further produce fracture to the fibula (usually avulsion) or to the tibia (usually due to a shift of the talus), producing an intra-articular fracture, (usually an oblique fracture involving the medial malleolus). Eversion of the ankle will produce trauma to the deltoid ligament or avulsion of the medial malleolus at its tip. Also fracture of the shaft of the fibula in a spiral manner may be seen. More severe injuries may fracture the tibia by a shift of the talus, but this is, fortunately, a rare occurrence.

Thus, the point to be made is that it is the x-ray that is diagnostic of the mechanism of the injury. There is no doubt that the patient's history is exceedingly important in the diagnosis of any problem in medicine, but at the same time there is great danger in believing everything a patient tells you and accepting it as correct. In this case the patient can't be right and must have sustained an eversion injury of the ankle in order to account for the x-ray finding of a fractured shaft of the fibula. Treating this as an inversion injury would not be the proper treatment. It could leave the patient with an unnecessary residual problem with respect to the stability of his ankle, increasing the deformity and leading to malunion of the fracture.

Normally, treatment of an ankle in which there has not been a fracture is directed toward resting the stretched ligaments. The common soft tissue injury is caused by inversion, and trauma to the lateral ligaments is treated by holding the ankle in *eversion* either by elastoplast tape or plaster of Paris. An eversion injury in which the medial deltoid ligament has been traumatzied is cast in neutral position.

TABLE 1

SUMMARY OF MAJOR INJURIES AT THE ANKLE JOINT

Stress	*Inversion*	*Eversion*
Mild	stretch ("sprained") lateral ligament	stretch ("sprained") medial ligament
Moderate	tear of lateral ligament	torn medial ligament
	avulsion of tip of fibular malleolus	avulsion of tip of tibial malleolus
		shearing fracture of fibular shaft
Severe	shearing fracture of medial malleolus (due to medial shift of talus) involving the distal articular surface of tibia	shearing fracture of fibular malleolus (lateral shift of talus)
		tibiofibular diastasis, with medial lig. tear, lateral shift of talus ± fractured shaft of fibula
	fracture of both medial and lateral malleoli with varied ligamentous injuries, ± injury of the posterior articular surface of the tibia due to talar shift or pull of the ligaments producing "the tri-malleolar fracture"	

If there has been a fracture, the mechanism of injury becomes clearer on x-ray, and treatment should be directed accordingly. Fractured distal malleoli do not involve the joint surface and thus may be reduced closed and casted with regard to the associated ligaments that might be strained or torn. Fracture of the shaft of the fibula as in this case may be treated closed by a below-knee cast, in the inversion position, which will hold the fibula in reasonable reduction, as well as resting the medial ligaments. This would include immobilization with no weight bearing for three weeks, followed by weight bearing for a further three or four weeks, depending upon the severity of the injury.

SUMMARY

This patient was seen to have sustained an eversion injury to his ankle which was diagnosed on x-ray. His injury was probably aggravated by associated ligamentous stretch or tear on the medial side of the joint. He was treated in a below-knee cast in the inverted position for a period of eight weeks. The fracture united, and he made an uneventful recovery with no residual instability or loss of function of the ankle. The point to be made here is that this satisfactory outcome may not have been accomplished had he been treated originally as an inversion injury on the basis of his history, and might have been left with a permanently weak deltoid ligament and thus an 'unstable' ankle.

Thus, although not the commonest injury seen in the ankle, this case illustrates well how a basic knowledge of the ankle movement mechanism may aid diagnosis and proper treatment of injuries at this joint.

ACKNOWLEDGEMENT

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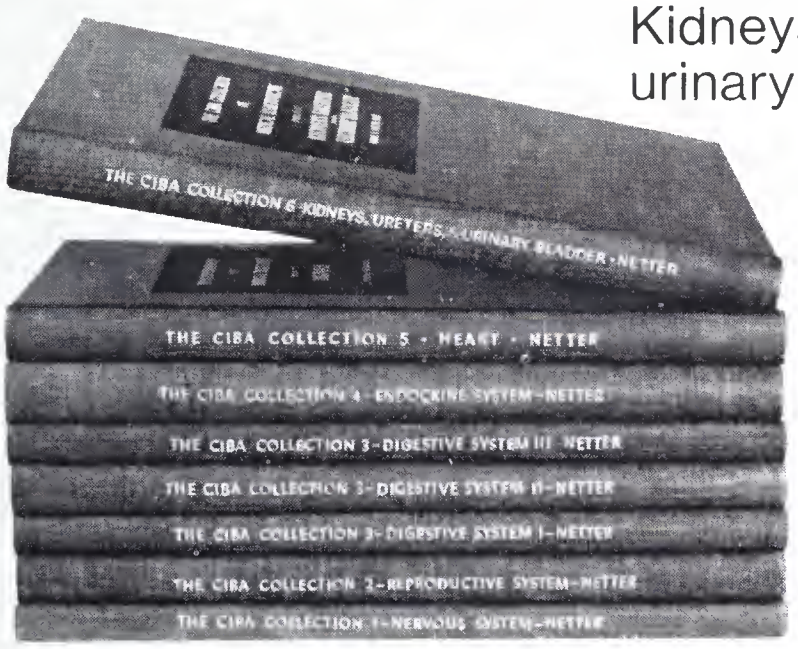
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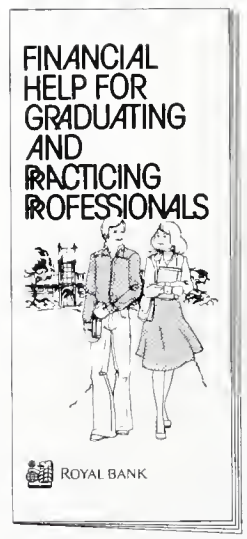

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